lipids as appropriate for each toxicant. Additional demographic and socioeconomic variables were retained as covariates in the models when statistically significant. We conducted sex-stratified analyses for each exposure-outcome association, determined a priori, regardless of measured interactions.

In multivariable analyses of individual toxicants, maternal BPA and phthalate concentrations were not significantly associated with any internalizing symptoms. Child reported anxiety and depression scores were elevated among children whose mothers had higher PBDE and PFC concentrations during pregnancy and among children with higher lead levels. Parent reported internalizing symptom scores were elevated among children whose mothers had higher PBDE and PFC levels. Parent reported social impairment scores were elevated among children whose mothers had higher levels of cotinine and among children with higher lead levels. Some associations varied by child sex. Additional analyses will clarify the roles of singular and multiple toxicant exposures in childhood internalizing symptoms.

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NTX50
Do peripheral inflammatory responses link early chronic low-level lead exposure and later psychiatric disease?

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Our studies have suggested that over half of minority children living in very low-income neighborhoods are chronically exposed to low-level environmental lead. Early chronic low-level lead exposure has been associated with poorer academic and neurocognitive function and neurobehavioral disorders. Few models have been proposed that suggest the causative pathways through which early chronic low-level lead exposure changes brain and behavior.

Lead exposure is associated with immune system dysfunction. Recently, neuropsychiatric studies have shown that abnormal proinflammatory immune responses are present in psychiatric patients with autism, depression and schizophrenia, as well as in those with a range of neurodegenerative diseases. Our laboratory has replicated and added to the literature on neurocognitive effects of low-level lead exposure in young children. In animal studies we have shown that, as compared with controls, C57BL6J mice chronically exposed to low-level lead had kidney glomerular hypertrophy and microglial abnormalities in dentate gyrus/hippocampus. Evidence from diverse research domains will be integrated to suggest a new model of early chronic low-level lead exposure in which changes in brain and behavior occur secondary to changes in organ function and peripheral immune response.

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NTX51
Neurotoxic effects on attention deficit and hyperactivity in rodent models

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Attention Deficit Hyperactivity Disorder (ADHD) is the most commonly diagnosed cognitive impairment of childhood. ADHD, as the name implies , is characterized by impairment of attentional function and locomotor hyperactivity. However, the symptoms of ADHD syndrome are multifaceted and also include impairments of behavioral control and disordered planning processes. There is considerable heterogeneity of symptoms within the diagnosis of ADHD. Some children primarily display attentional impairment without hyperactivity, while others exhibit both, and the ancillary symptoms may be present to greater or lesser extents. Neurotoxic exposures during development have been found to be associated with attentional impairment and higher rates of ADHD. In particular, maternal tobacco smoking has repeatedly been found to be significantly associated with higher ADHD rates in offspring. Animal models are useful for defining the direction of the causative arrow. We have recently shown that developmental exposure of pregnant rats to tobacco smoke extract (TSE) causes significant locomotor hyperactivity in the offspring. Impairment in novel object recognition, a low motivation test of attentional function, is also seen in the TSE offspring. In contrast, during the operant visual signal detection task, an appetitive, high motivation test of attentional function, the TSE offspring do not show impaired performance. Other toxicants such as heavy metals and pesticides have also been found to cause attentional impairments and locomotor hyperactivity after early life exposure. Animal models can provide key causative and mechanistic information concerning the relationship of early life neurotoxic exposure and long-term ADHD-like dysfunction.

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NTX52
Early life lead exposure and schizophrenia neuropathology: Effects on parvalbumin-positive GABAergic interneurons and subcortical dopaminergic activity

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Environmental factors have been associated with psychiatric disorders and recent epidemiological studies suggest an association between prenatal lead ($Pb2+$) exposure and schizophrenia (SZ). $Pb2+$ is a potent antagonist of the N-methyl-D-aspartate receptor (NMDAR) and converging evidence indicates that NMDAR hypofunction plays a key role in the pathophysiology of SZ. The glutamatergic hypothesis of SZ posits that NMDAR hypofunction results in the loss of parvalbumin (PV)-positive GABAergic interneurons (PVGI) in the brain. Loss of PVGI inhibitory control to pyramidal cells alters the excitatory drive to midbrain dopamine